

The Case | Milky ascites is not always chylous

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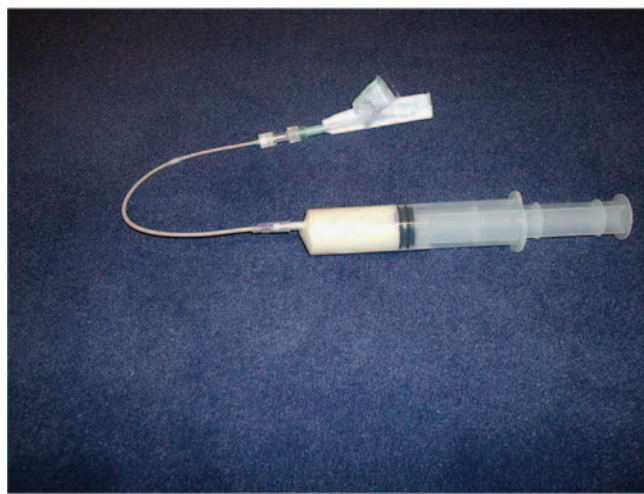


Figure 1 | Milky ascites.

A 57-year-old woman was treated with peritoneal dialysis for 15 years, after which ultra-filtration failure occurred. Permeability testing of the peritoneum was suggestive for sclerosing peritonitis. She was switched to hemodialysis. Half a year later she developed clinically manifest ascites. Diagnostic paracentesis revealed milky ascites resembling

chylous ascites (Figure 1). However, even after appropriate dilutions no triglycerides were detected. Further evaluation of the ascites was consistent with an exudate, pH 7.54 containing 2.42 mmol/l calcium and 5.58 mmol/l phosphorus. Serum calcium and phosphate levels were normal; parathyroid hormone (PTH) level was 42 pmol/l.

What is the cause of this patient's milky ascites?

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The Diagnosis | Calcifying sclerosing peritonitis

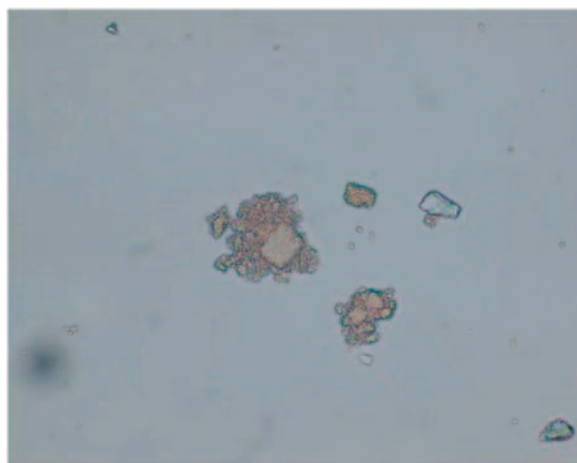


Figure 2 | Calcium-hydroxyphosphate-apatite crystals.

Because of the milky aspect of the fluid further tests were done, which showed calcium-hydroxyphosphate-apatite crystals (Figure 2) and massive amounts of colloidal calcium-phosphate-fetuin-A complexes, or calciprotein particles (CPPs).

Calcifying sclerosing peritonitis is a particularly severe form of dialysis-associated calcification.¹ Tissue calcification in chronic kidney disease (CKD) patients is driven by an imbalanced mineral homeostasis. The elevated calcium and phosphate concentrations result in a chemical precipitation reaction that can lead to mineral deposition in the extracellular fluid (calcium-hydroxyphosphate-apatite crystals). Other calcification inducers in CKD patients are an increased PTH level and excessive treatment with vitamin D.² A number of calcium-regulatory factors control and prevent unwanted extra-osseous calcification: Matrix Gla protein acts locally, for example, with the elastic lamellae of arteries preventing media calcification, while pyrophosphate and the plasma protein fetuin-A act systemically in blood and tissue fluids. Fetuin-A is a liver-derived plasma protein that has the ability to prevent the precipitation of basic calcium phosphates from supersaturated solutions by the two-step formation of CPPs, which may serve as a transport form of otherwise insoluble mineral debris. Acidic plasma proteins,

including albumin, greatly stabilize CPPs after the initial formation.

In general, dialysis patients often suffer combined deficiency in fetuin-A and albumin, which can explain why these patients are at a much higher risk of calcification and associated cardiovascular morbidity and mortality.³

Although our patient had a well-controlled calcium-phosphorus product, the presence of high inflammatory activity during her sclerosing peritonitis may have evoked the precipitation of the calcium-hydroxyphosphate-apatite crystals by lowering the serum fetuin-A, which is a negative acute-phase protein.

She was treated with tamoxifen while her calcium-phosphorus levels were further improved. At 2 years of follow-up, she remains well with a stable amount of ascites.

REFERENCES

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